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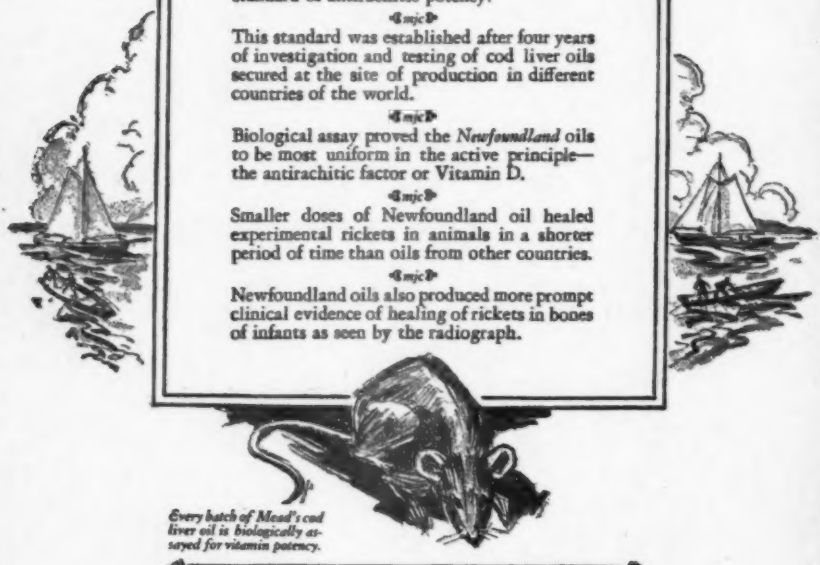

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THE DEVELOPMENT OF THE MENINGES IN AMPHIBIA

A Study of Normal and Experimental Animals. Preliminary Note[1]

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It has been pointed out by Kölliker, His, Salvi, Sterzi, Weed, van Gelderen, and Clermont that the meninges of man, pig, mole and certain of the fishes all come from the middle germ layer. The observation was made by Weed that the first appearance of a subarachnoid space is correlated with the beginning of extra-ventricular spread of cerebrospinal fluid. Weed suggested, in consequence, the possibility that the cerebrospinal fluid is an exciting agent in meningeal differentiation: the fluid, passing outward from the cerebral ventricles, cuts for itself a channel in the perimedullary tissues and thus forms a subarachnoid space. The last notable addition to the problem was offered by the work of Harvey and Burr. These investigators, seeking an explanation for the differences exhibited by the pachymeninx and leptomeninges in their reaction to and recovery from trauma, came to the conclusion, by means of neuro-transplant experiments, that the leptomeninges arise from mesenchyme which has as its precedent neural crest cells, whereas the pachymeninx arises independently of the crest cells. This hypothesis, beyond its embryological significance, makes possible a new approach to the pathological problems of the meninges.

A study of the comparative embryology and anatomy of the meninges brought no substantiation of the belief of Harvey and Burr. Furthermore, the neural crest shifts so as to leave largely entodermal mesenchyme in the dorsal head regions and to give for the most part ectodermal mesenchyme in the ventral head regions. The question immediately suggests itself: From what does the pachymeninx develop ventrally and what gives rise to the leptomeninges dorsally? These

[1] Received for publication October 15, 1927.

questions combined with the thought that the method of transplantation gives an excellent means of experimentally determining the influence of fluid in the differentiation of the meninges led to a repetition and extension of the work of Harvey and Burr.

Normal embryos of *Amblystoma punctatum* were first studied. The process of meningeal development here is strikingly analogous to that described by Weed for pig and man. In the youngest embryos, there exists an area (in Weed's terminology, the *area membranacea ventriculi quarti*) differentiated to permit the passage of fluid from the roof of the fourth ventricle to the periaxial tissue. The first cleavage in this perimedullary tissue to give an embryonic subarachnoid space occurs shortly after the appearance of chorioid plexus cells; the maximum rate of development of the space is seen in stages where the chorioid is developing most rapidly. The pia mater develops from the inner layer of the mesenchyme; the dura, by fibrosis of the outer layer. The innermost shell of cells lining the dura escapes the fibrotic process and, by separation from the fibrous dura beneath it, establishes the arachnoid membrane.

It was found to be of importance to study the types of cells present in the dural and pial condensations, for herein lies the true test of the validity of the views of Harvey and Burr. The picture is an indubitable one: side by side in the lateral portions of the condensations there are large, light-staining mesodermal cells with much yolk; small, dark-staining neural crest cells with scanty cytoplasm and little yolk; and mesodermal cells which have lost the characteristics that permit identification of their origin. It is impossible, in consequence, to reconcile the experimental findings of Harvey and Burr with the cell-relations in the normal animal.

In the experimental series, two groups of transplants were made with spinal cord; one with, the other without, neural crest cells. All of these transplants, ten in number, after about two months in the host, develop a pial covering, complete in some instances, incomplete in others. None of them develops a definitive subarachnoid space, arachnoid membrane, or dura. The presence or absence of neural crest cells in no way influences the formation of membranes about the transplanted cords. In so far as all but ependymal secretion is absent and since there is no avenue of escape for the small amount of spinal

fluid present, no formation of definitive membranes is to be expected if fluid be supposed to be the exciting agent in cleavage of the peritransplantal tissue.

Two series of transplants were made with brain, paralleling those made with spinal cord. All of the specimens in this brain series acquire a well formed pia mater and all have a chorioid plexus. Four of the transplants (two with, two without neural crest cells) have about them dura and subarachnoid space remarkably like those surrounding the normal central nervous system. In all of these instances, the formation of a fluid-channel and a pachymeninx is correlated with a peritransplantal fluid-spread. Five of the transplants (three without, and two with neural crest cells) are without membranes other than the pia mater. Nor is membrane formation, in these instances, to be expected on the basis of fluid-passage. The presence of neural crest cells has failed to lead to observable differences in the meninges of the transplants. Fluid-escape into the peritransplantal tissue, however, is correlated with the presence of a subarachnoid space and definitive meninges.

A full account of the observations and experiments briefly epitomized here will appear as Contribution 110, Volume 20, "Contributions to Embryology," Carnegie Institution of Washington.

AN EXPERIMENTAL INVESTIGATION INTO THE EFFECTS OF ASPHYXIA ON THE BRAIN, WITH ESPECIAL REFERENCE TO ASPHYXIA NEONATORUM[1]

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INTRODUCTION

In 1861, Little's paper, "On the influence of abnormal parturition, difficult labors, premature birth, asphyxia neonatorum, on the mental and physical condition of the child, especially in relation to deformities" (1), was presented before the Obstetrical Society of London. The group of spastic palsies which Little discussed included those due to spinal cord injuries, hemiplegias, monoplegias and diplegias, and he mentioned various etiological factors, as the title of the paper indicates. However, Little apparently attributed the diplegias more specifically to asphyxia and concluded from the studies of Weber and Hecker that the anatomical basis consists of congestion and petechial hemorrhages, or as he terms it "capillary apoplexy." Since Little's time no additional evidence has been offered to support his views, but they are still widely accepted and expounded in most textbooks of obstetrics, neurology and pediatrics. For example, in the recent publication of Hausman and Sachs (2) the statement is made that "asphyxia neonatorum with its meningeal hemorrhages" is often responsible for cerebral birth palsies.

It was the purpose of this investigation to determine whether asphyxia alone may leave permanent brain damage, or more specifically, whether asphyxia neonatorum may play any part in the etiology of the infantile cerebral palsies.

OBSTETRICAL EVIDENCE

Asphyxia neonatorum may be caused by any accident which prevents the initiation of pulmonary respiration immediately after the

[1] Received for publication November 18, 1927.

placental circulation has been interrupted. For example, the cord may be twisted, knotted or compressed during delivery, the placenta may be prematurely detached or the infant, for various reasons, may fail to breathe on delivery. It is important to realize that intracranial hemorrhage from birth trauma is a common reason for failure to breathe. This condition is the so-called "pallid asphyxia" and is the result not the cause of intracranial hemorrhage.

Most of the older writers have ascribed gross cerebral hemorrhages at birth to asphyxia, but as a result of many careful studies of the mechanical factors involved it is now being generally recognized that such gross lesions are due to trauma. Beneke (3), Holland (4), Ehrenfest (5), Pierson (6) and many others have shown that moulding of the foetal head may cause rupture of the dural septa with tearing of the venous sinuses and smaller vessels. The vein of Galen may be put under tension and the vein itself or one of its tributaries ruptured. The tributaries of the longitudinal sinus are sometimes torn by an over-riding of the parietal bones. In the opinion of the best obstetrical writers of today large intracranial hemorrhages in the newborn are amply accounted for by mechanical injury and are never due to asphyxia alone. There is no doubt, however, that asphyxia may increase the bleeding from a torn vessel or play a part in the rupture of abnormally fragile vessels.

If we exclude gross intracranial hemorrhages as a result of asphyxia, we must, nevertheless, admit that multiple petechial hemorrhages in the brain and meninges are found in all forms of asphyxia, not only in infants but also in adults. There seems to be no clinical evidence that such petechiae are of any importance. A more pertinent question, however, remains to be answered, namely, whether it is possible for an animal to survive a degree of asphyxia which is severe enough to cause necrosis of the nerve cells. In this connection Crothers (7) has recently referred to the experiments of Pike and others (8) on the interruption of the cerebral circulation in cats in which it was found that the medullary centers are more resistant to anemia than the cerebral cortex, and that animals may survive a cerebral anemia prolonged to the point of causing necrosis of the cortical cells. Severe lesions in the brain are found after poisoning by illuminating gas and are commonly ascribed to the action of the carbon

monoxide that it contains. As physiologists have shown that carbon monoxide has no direct toxic action on nerve cells and that carbon monoxide poisoning is merely an anoxemia, it would seem reasonable to suppose that other types of anoxemia of appropriate depth and duration would cause similar lesions in the nervous system. This problem will be discussed at greater length in the next section.

The clinical evidence is, on the whole, against Little's theory. This evidence has been analyzed in a previous paper (9). Suffice it to say here that clinical studies by Manton (10), Bruckhardt (11), Beatus (12), Hannes (13), Kwozek (14), Rechschaft (15) and Hannes (16) have afforded no support to the theory that asphyxia alone is an important factor in cerebral birth injury. Of course, the problem can never be solved on clinical grounds, because of the impossibility of excluding traumatic lesions.

CARBON MONOXIDE POISONING

It is well known that poisoning by illuminating gas may produce diffuse and focal lesions in the brain including, characteristically, necrosis of the anterior third of the globus pallidus. These lesions are generally ascribed to the carbon monoxide contained in illuminating gas and numerous case reports have been published along these lines. Neuropathologists commonly explain the focal necroses as due to a selective action of carbon monoxide on the globus pallidus. On the other hand, Haldane (17) and other physiologists have shown experimentally that carbon monoxide has no directly toxic action on the nerve cells, but produces its effects only by its strong affinity for hemoglobin from which it displaces oxygen and thus prevents oxygenation of the tissues. Haldane has been able to keep mice alive when exposed to from 200 to 300 times the fatal concentration of carbon monoxide by increasing the oxygen tension so that enough oxygen could be dissolved in the plasma to supply the tissue needs, although the hemoglobin was completely saturated by carbon monoxide. Haggard (18) has recently furnished a possible explanation for this difference of opinion. He showed that tissue cultures of embryonic nerve cells could be grown successfully in an atmosphere of 79 per cent carbon monoxide, but that even a minute trace of illuminating gas

was sufficient to kill the culture. It is probable, therefore, that illuminating gas contains some substance other than carbon monoxide which is at least in part responsible for the injury to the brain. The effects of pure carbon monoxide on the brain have not yet been described.

EXPERIMENTAL WORK

A large number of cats and kittens were asphyxiated in various ways. Kittens were soon found to be more suitable and were used exclusively in all later experiments. In the first series of experiments the animal was asphyxiated rapidly by washing out the air from a bell jar with nitrogen. Resuscitation was delayed as long as possible. After respiration had failed there was an interval of three to five minutes during which artificial respiration was found to be effective, but after this period the heart stopped and could not be started in most cases, although epinephrine and other cardiac stimulants were injected directly into it. If the animal were revived, it always recovered promptly and completely. Four kittens from three litters were asphyxiated severely and their four siblings were kept as controls. No difference could be made out in the weight, rate of development or general activity of the two groups. Seven animals were killed after asphyxiation, the time varying from immediately after the conclusion of the experiment to two weeks later. In no case could any gross or microscopic hemorrhage be found, but extreme congestion was detected in cats killed soon after asphyxiation. Tissues were fixed in formalin and alcohol and stained with hematoxylin and eosin, by Nissl's method, with Sudan III and Scharlach R, and with Weigert's myelin stain. No gross or microscopic lesions were found which could be attributed to the asphyxia except the transient congestion, and this disappeared after a few hours. It was concluded that anoxemia brought the heart to a standstill before any lasting damage was inflicted on the nervous system. This experiment was designed to duplicate the sudden more or less complete type of asphyxia which might occur during birth. Since the results were negative, it was planned to try the effects of prolonged low oxygen tension, in order to discover whether any type of asphyxia might cause brain injury,

although the results might not be directly applicable to the problems of birth injury.

In this second series of experiments kittens were placed in a bell jar and a constant stream of a low oxygen mixture was run through. In this way kittens could be kept for as long as 12 hours in an atmosphere of 5.5 to 6 per cent oxygen. The animals could not be kept alive for more than a very short while when the oxygen was below 5.5 per cent. In successful experiments the kitten would be cyanotic and dyspneic and unable to stand or hold up its head for many hours. Most of the time the animal would be drowsy, but would always rouse at times and was never deeply comatose. After prolonged dyspnea the animal would often collapse suddenly so that immediate resuscitation would become necessary. When revived, the kitten would be unable to stand for half an hour or an hour and would yowl for some time. For some hours it was weak and tremulous, but usually seemed normal on the next day. If the animal were asphyxiated only once, its nutrition did not suffer, but repeated severe asphyxiation for prolonged periods caused interference in development and the kitten would be definitely smaller and weaker than its siblings. The brains of five kittens were studied. Tissues were fixed in formaline and in alcohol and stained with hematoxylin and eosin, by Nissl's method, with Sudan III and Scharlach R, and with Weigert's myelin stain. No gross or microscopic lesions were found.

For comparison a few cats were asphyxiated with carbon monoxide. The animal was first rendered unconscious by a strong concentration of carbon monoxide and then placed in a bell jar and a stream of 0.3 per cent carbon monoxide run through. This method was described by Cobb and Forbes (19). Under these conditions the cat might be kept unconscious for several hours, even deeply comatose. However, the next morning it would show no definitely ill effects or might be slightly weak or sluggish. No lesions were found in the brain in two cases. However, when illuminating gas was employed in one experiment, the cat was unable to walk and was apparently blind until it died on the third day. A second kitten poisoned by illuminating gas was comatose for six hours and showed severe nervous disturbances until killed on the sixth day. It was tremulous and ataxic, could not stand, walk, or drink milk. Grossly, the brain was unaltered, but

Nissl's cell stain showed chromatolysis of many cells in the basal ganglia, and scattered cells in the cortex were also altered. The changes were best shown by the fat stains which revealed fat accumulations and degeneration of the nerve cells in the lenticular nucleus and less severe changes in the cortex. The small blood vessels in the basal ganglia were also affected. There were areas where the endothelium was swollen and contained many fatty granules.

CONCLUSIONS

1. It is still widely taught that asphyxia neonatorum is a common cause of cerebral birth injury, although the best clinical studies by Hannes and others lend no support to this theory.

2. The writer was unable to produce brain lesions in cats and kittens by the employment of various types of experimental asphyxia.

3. Cerebral lesions were not produced in two experiments with pure carbon monoxide, but when illuminating gas was used, the well known lesions were found. Hence, it seems probable that carbon monoxide alone is not responsible for the cerebral lesions which result from illuminating gas poisoning.

I wish to thank Professor Adolf Meyer for his kind interest and advice.

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A STUDY OF THE CONVULSIONS PRODUCED BY WORMWOOD[1]

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INTRODUCTION

The enormous literature bearing on the problem of epilepsy and convulsive phenomena in general records many investigations on experimental convulsions in animals; the conclusions arrived at in such studies have been applied to the problem of epilepsy in humans, many times without justification in fact. Contemporary research into the question of convulsive seizures centers mainly about three methods: (1) convulsions following transient cerebral anaemia, (2) convulsions from electrical stimulation of the cortex, and (3) convulsions produced by the action of drugs.

Elsberg and Stookey (1923) clamped the innominate and left subclavian arteries in cats, thereby producing a cerebral anaemia. After reestablishment of the circulation, tonic and clonic convulsions ensued. They observed that one convulsion was followed by a lessened susceptibility to the seizures; this raised threshold lasted for one to three days and was regarded by them as an exhaustion phenomenon. They also showed that if, during the period of cerebral anaemia, the cerebral vessels were perfused with Ringer-Locke or normal saline solutions, no convulsions ensued on reestablishment of the cerebral circulation. These results cannot be directly applied to the findings in epilepsy, inasmuch as an anaemia of the brain in epileptic attacks has not been demonstrated and a cerebral anaemia from vasomotor spasm is still an occurrence of doubtful possibility. Because of the generalized character of the cerebral anaemia produced by such a method, nothing definite may be said as to the origin of the convulsions and no localization of function in the mechanism is determined.

Electrical stimulation of the cerebral cortex has been often employed

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to produce convulsions. The results obtained by this method have been cited to prove the cortical origin of epileptic seizures. Luciani (1915), in a lengthy summary of the older work on the subject, supported the theory that the motor phenomena of convulsions have their origin in the cerebral motor cortex and that, in the cases of convulsions produced by stimulation of a sensory area, a diffusion of the stimulation to the motor area must have occurred. The fact that bilateral convulsions have sometimes been seen after the motor cortex has been destroyed on one side was explained on the basis of a diffusion of the excitation to motor centers of the bulb, which "may be considered as accessory and complementary although indispensable factors."

Dandy (1927), working with acute preparations, recently found that clonic convulsions occurred only on stimulation of the cerebral motor cortex or its subjacent fibers with the motor cortex intact; that after unilateral extirpation of the motor cortex, clonic convulsions never developed contralaterally; and after excision of the motor cortex of both hemispheres, clonic convulsions were not again seen regardless of the point of stimulation or the intensity of the stimulus. He concluded: "The motor cortex is alone responsible for the remarkable phenomenon of clonic convulsions."

Probably the most widely used method of producing convulsive seizures in animals is that of introducing some drug into the circulation. Of these, acid fuchsin and wormwood, the active ingredient of absinthe, have been most extensively employed. Barbour and Abel (1900) found that convulsions in frogs were readily induced by acid fuchsin and noted a greater susceptibility after a slight injury to the brain. It was observed later (Abel, 1912) that after such an injury the central nervous system became diffusely infiltrated with the dye. The former observers also showed that a greater susceptibility to convulsions from the dye was present in cardiectomized than in normal frogs and attributed this lowered threshold to asphyxia. This work was entirely corroborated by Syz (1923) who later concluded (1926) that asphyxia increased the convulsive action of acid fuchsin and in some way caused a greater absorption of the dye into the central nervous system. Sauerbruch (1913), working with rabbits and monkeys, obtained results comparable to those of Abel and Barbour, in frogs.

Wormwood as a convulsant drug was first employed by Morcé

(1864), and since its endorsement by Hughlings Jackson and Victor Horsley has been commonly used for this purpose. The seizures following its administration were believed by these famous observers to give the nearest likeness to the clinical picture of epilepsy.

Just how the drug acts has never been determined. The opinion of the older writers has been summarized by Luciani (1915); "Some poisons, particularly absinthe, when introduced into the circulation, produce convulsions similar to those initiated by electrical stimulation of the cortex. These attacks are also seen in animals in which the brain-stem is completely severed from the brain. Injection of a few drops of tincture of absinthe produces inexcitability of the cerebral cortex with simultaneous onset of violent epileptic seizures due to the excitation of bulbo-spinal centers."

The increased susceptibility of epileptics to convulsions has been thought by some observers to be due to focal damage to the central nervous system, by others to be due to changes in the cerebro-spinal pressure. The former phase of the question has been investigated by Pike and Elsberg (1925), who found that convulsions occurred after the administration of an alcoholic solution of absinthe intravenously from one to three weeks after extirpation of the motor cortex on one or both sides. They reported that in such preparations the dosage of the drug required to produce a convulsion was somewhat greater than in the normal animal. There was greater tonicity and extensor spasm on the injured side and the animal rolled toward the side of the uninjured hemisphere. These workers believed that the cerebral motor cortex was unnecessary for the production of those recurrent muscular contractions which occur in a typical convulsive attack due to absinthe. This is contradictory to the evidence obtained from electrical stimulation experiments as to the rôle of the cerebral motor cortex in the mechanism of the convulsion (Dandy).

Dandy and Elman (1925) made a further contribution to the subject when for the first time they administered the drug to cats with old healed lesions of the central nervous system. They produced minor injuries to the brain, of no uniformity in character or location, which, it was thought, would have some analogy to the brain defects so often found in epileptics at operation or necropsy. The drug was administered by stomach tube as an emulsion in gum acacia solution. They

found invariably a hypersusceptibility to convulsions from three to seven times greater than in normal cats, judging susceptibility by the dosage of the drug just necessary to produce convulsions. Injuries to the cerebral motor cortex caused the greatest increase in susceptibility, while injury to other parts of the brain produced less marked changes.

Leriche and Wertheimer (1925), however, concluded from their experience with cases of Jacksonian epilepsy following war wounds, that neither foreign bodies in the brain nor meningeal lesions were responsible for convulsions, but that pressure changes in cerebro-spinal fluid were the causative agent.

Elsberg and Pike (1926) investigated the rôle of cerebro-spinal fluid tension in the production of convulsions and found that cats given absinthe intravenously were more susceptible to convulsions when the pressure of cerebro-spinal fluid was greater than normal and less susceptible when the pressure of the fluid was less than normal.

In this paper the relation of chronic injuries of the central nervous system to the minimal dose of absinthe required to produce a convulsion in cats will be considered. The work reported is an amplification of other recent experiments. For it seems important to study further the relation of cerebral injuries to convulsive seizures. A number of cats have been utilized; in one group an extensive injury of the motor cerebral cortex was produced, in another minor lesions, while in still other animals the effect of spinal injuries was observed. The motor cortex was stimulated in eight cats after the administration of absinthe to test the effect of the drug on the irritability of the cerebral motor area. These latter experiments proved in many ways the most interesting of all.

METHODS

Eleven animals subjected to cerebral operations were studied in these experiments; another group of seven normal animals was used as controls. The operations were all performed with aseptic technique. An incision was made in the scalp through the dorsal mid-line and after the superficial fascia and musculature had been reflected, an opening in the bone was made with a trephine. This opening was enlarged with rongeurs; bone bleeding was controlled by the application of wax. The dura was then opened over the area of cortex to be removed; the

actual incision was made with a small knife or in some cases with the cautery. The wound was then closed; no cerebro-spinal fluid leak ever occurred. The wound in every case healed well and the animals remained in good condition. No spontaneous convulsions were ever noted in the operated group.

After a number of weeks had passed the drug was administered to these cats. The oil of wormwood was given by stomach tube in a 5 per cent suspension in gum acacia. This method had already been utilized by Dandy and Elman with good results.

OBSERVATIONS

A. Description of a typical wormwood convulsion and determination of the minimal convulsive dose

Five or ten minutes after administration of the drug, definite prodromal symptoms were seen. The animal became increasingly irritable to external stimuli and involuntary movements of the extremities appeared.

The convulsion was initiated by a sudden falling of the cat to the floor with loss of consciousness; during the convulsion the animal was insensible to external stimuli, such as touch and loud sounds. Marked tonic contraction of the muscles ensued,—flexion of the neck and forelegs, fixation of the jaws, extension of hind-legs and full extension and wide separation of digits. After a few seconds the tonic phase gave way to the clonic; a marked extension of the neck occurred with clonic movements of the jaws and violent clonic beats of the fore- and hind-legs. Later the movements of the legs became less violent, resembling more closely typical running movements. With the first tonic contraction of the muscles a peculiar crowing was often heard due to forced expiration past adducted cords; during the seizure respirations ceased. The clonic convulsions gradually became less forceful and ended in one to three minutes.

The convulsion was followed by a period of variable length during which the cat was comatose, and insensible to ordinary external stimuli. During this stage the muscles were completely relaxed. The animal then slowly regained normal behavior, although it was weak. Parts of the attack other than the convulsion proper might be reenacted but if the dose were minimal no further convulsion ensued.

Seven normal cats were given the oil of wormwood emulsion by stomach tube to determine the minimal dose of the drug required to produce a convulsion. The dosage in cubic centimeters of the drug per kilogram of body-weight was arrived at after several administrations to each of the animals. On averaging the minimal dosage for these seven cats it was found to be 2.08 cc.

B. Administration of wormwood to cats with small injuries of the motor cortex

Dandy and Elman (1925) found that cats with small injuries to the cerebral cortex became after a number of weeks more susceptible to the drug, convulsions following its administration in smaller doses. Their results were confirmed in this work by a study of three animals in which a fine puncture wound was made unilaterally in the region of the motor cortex from five to fourteen weeks before administration of the drug. The average dose by kilogram of body-weight required to produce convulsions in these animals was 0.92 cc. This minimal dose, then, is distinctly lower than that required in normal cats (2.08 cc.); the ratio is 1:2. The findings then suggest, as did those of Dandy and Elman, that a healed minor lesion of the cortex forms a locus minoris resistentiae. The increased susceptibility to the drug, however, was not as striking as in Dandy and Elman's experiments.

The animals with cortical injuries showed in the character of the convulsions alterations which seemed definitely related to the nature of the injury. In the cats with minor cortical injuries, the prodromal signs and symptoms of the convulsion were markedly reduced or altogether absent except for increased excitability to touch and to loud noises. The cats often appeared practically normal and then suddenly fell down unconscious in a violent convulsion. There was no unilateral weakness present. The attack was of short duration but included both a tonic and clonic phase. Moreover, it was not followed by that exhaustion seen in the normal animals; recovery was almost immediate.

C. Administration of wormwood to cats with large lesions of the motor cortex

At the time that these experiments were begun, a number of adult cats with extensive lesions of the motor cortex were available in this laboratory. Observations on the behavior of these animals have already been reported by King (1927). These cats are of particular interest in that the exact portion of the motor area removed was determined both at operation and at autopsy.

When the cerebral cortex of adult cats is stimulated by means of a unipolar electrode and weak current, the motor area is found to surround the cruciate sulcus (Weed and Langworthy, 1926). On stimu-

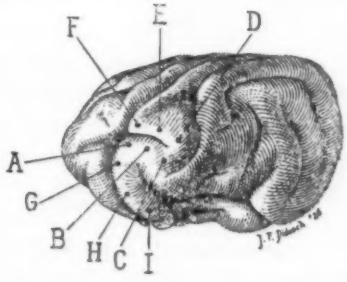

FIG. 1. DIAGRAM OF THE LEFT CEREBRAL CORTEX OF AN ADULT CAT.

lation of areas *A* and *B* (Fig. 1) a contraction of the contralateral foreleg muscles was obtained; from areas *E* and *F*, a contraction of the contralateral hind-leg. Stimulation of area *C* (Fig. 1) caused a contraction of the facial and masticatory muscles of the opposite side. Within these areas smaller centers were often located which gave on stimulation more circumscribed reactions, but these smaller centers showed great variation in the individual animals.

King (1927) removed portions of the electrically responsive motor cortex either unilaterally or bilaterally in a number of cats at aseptic operation and also removed the cortex situated frontally to this responsive area (areas *G*, *H* and *I*, Fig. 1), including also a portion of area *A*. Removal of the motor area resulted in no great paralysis of

the extremities but rather in a transient weakness which disappeared after forty-eight hours. Removal of areas *A*, *G*, *H* and *I*, on the contrary, gave rise to abnormalities in postural tone seen as extensor hypertonus of the contralateral extremities.

Six animals with extensive lesions of the motor cortex were utilized in these experiments in order to learn the minimal dose of oil of wormwood required to produce a convulsion. The results are seen in Table I; by referring to Figure 1 it is possible to localize accurately the portion of the motor cortex removed in each of the animals. The figures in parentheses represent the number of weeks that elapsed between the operation and the time the drug was given, a period of from five to eleven weeks.

TABLE I

NUMBER	POSITION OF LESION	MINIMAL CONVULSIVE DOSAGE
		cc./kgm. body-weight
1	Left cortex D. E. F.	2 (10)
2	Bilateral A. G. I.	3 (6½)
3	Left cortex A. G.	3 (7)
4	Left cortex entire motor area	3 (11)
5	Bilateral D. E. F.	3 (5)
6	Left hemisphere	3 (6½)

Average dose in cc./kgm. body-weight = 2.85 cc.

It will be observed that in these animals the average minimal dose of the drug required to produce a convulsion was 2.85 cc. per kilogram of body-weight. Since the normal cats examined showed convulsions with a smaller dose of the drug (2.08 cc.) it would appear from these experiments that animals after removal of extensive portions of the motor area were somewhat less susceptible to convulsions than normal animals: the ratio is roughly 3 to 2. It is interesting to note, then, that after destruction of an appreciable portion of the motor cortical area the susceptibility to convulsions is lessened. This is in accordance with the results of Pike and Elsberg (1925) and serves to correlate their findings with those of Dandy and Elman (1925).

In these cats with large cortical extirpations, the prodromal period of the convulsion was always prolonged and severe. The convulsion

itself was violent and the succeeding exhaustion very marked. This may all be attributed to the excessively large dose required to produce the convulsion. The side of the body contralateral to the site of the cortical injury was always weak and the animal fell to that side consistently. After the convulsion it walked in small circles to the weak side. The convulsions in every cases were bilateral even in the animal with one entire hemisphere removed.

It seems important to emphasize the fact that in each of the animals the convulsions were bilateral and generalized; a unilateral convulsion was never observed nor one in which any extremity failed to participate.

D. Administration of wormwood to cats with injuries to the spinal cord

The experiments of Dandy and Elman (1925) show not only that small injuries to the motor cortical areas make the cats hypersensitive to absinthe, but also that injuries in other portions of the central nervous system had this same effect although to a less degree. Thus they found that lesions in the cerebellar cortex made the cats react to smaller doses of the drug.

In the present study spinal cord injuries were produced in two cats. In one animal the dorsal and ventral spino-cerebellar tracts were cut on one side; in the other, the posterior column fibers and also the spino-cerebellar fibers were severed in the mid-dorsal region. Marked ataxia of the hind-legs was noted for the first three weeks after the operation; then this ataxia almost completely disappeared. When these cats were given oil of wormwood from four to five weeks after the operation, it was found that convulsions occurred with smaller doses than in normal animals. The average minimal dose in these cats was 1.22 cc. per kilogram, almost half that of normal cats.

At the time when the reactions of these animals to wormwood were tested, they had apparently almost recovered from the ataxia that immediately followed the spinal cord injury. Under the influence of the drug this incoordination of the extremities was found to return to a very marked degree. In the stage of excitement that preceded the convulsion the ataxia of the hind-legs was as great as it had been immediately after the operation and the gait of the cat was grossly

incoordinated. The incoordination was seen to return in one cat fifteen months after the spinal operation. This return of ataxia seems to have the same significance as weakness of the extremities on one side in those animals with contralateral motor cortical injury. Apparently the new mechanism of control is less secure than normal and succumbs easily after administration of the drug.

E. Stimulation of the cerebral motor cortex in cats after administration of wormwood

The mechanism of the convulsion after wormwood administration is not clearly understood. It has been thought that the attack is due to a stimulation of the bulbo-spinal centers freed from the control of the cerebral motor cortex which has been rendered inoperative by the drug. To test this theory the cerebral cortex was stimulated electrically after administration of the oil of wormwood.

In the eight experiments recorded here the cats were anesthetized and the cerebral motor cortex exposed bilaterally. Under light anesthesia the motor cortex was stimulated with a unipolar electrode, a fine platinum wire mounted on a spiral spring. The large indifferent electrode was attached to the side of the body. A current was found which was just sufficient to elicit contraction of a single muscle group, flexion of a leg, or of the facial and masticatory musculature on the contralateral side. Relaxation of the muscles occurred immediately on removal of the electrode. In no case did clonic contractions ensue and the contraction was always well localized.

The cats were then given 5 per cent oil of wormwood in gum acacia solution by stomach tube in a quantity which experience showed was a subliminal convulsant dose. Then with the same strength of current and continuance of the anesthesia, the motor cortex was again stimulated. A contraction of a muscle group now occurred which was followed, after the electrode was removed, by clonic contractions of the affected parts. For example, the fore-leg center in the motor cortex was stimulated. A sharp contraction of the contralateral fore-leg occurred which was not relaxed upon removal of the stimulus but continued as pawing movements of the extremity.

Moreover, the convulsion spread from a clonic contraction of the facial muscles on removal of the stimulating electrode to involve first

the fore-leg and then the hind-leg until a complete unilateral convulsion ensued. This unilateral convulsion was in turn invariably followed shortly by a generalized clonic convulsion, the spread to the opposite side being general and immediate and not following any particular sequence. Moreover, after administration of absinthe, stimulation of the sensory projection area posterior to the motor area gave a strong motor response. These findings indicate that after a subliminal dose of the convulsant drug the cerebral motor cortex is hyperexcitable. The added stimulus of electrical stimulation precipitates a convulsion even after a subliminal dose of the drug.

The strength of electrical stimulus that must be applied to the cerebral cortex in order to produce a response varies somewhat in different animals and in a single experimental preparation during the period of observation. If the motor area is continually stimulated with a current just strong enough to cause a response, fatigue sets in and no reaction can be obtained. If then administration of the drug renders the cortex again responsive to this same strength of stimulus, it seems clear that the irritability of the cortex has become increased.

Other cats were subjected to similar procedures except that the amount of wormwood administered exceeded the convulsive dose. In the prodromal period the motor cerebral cortex was again found to be hyperexcitable and clonic movements of the muscle groups persisted after the stimulus giving rise to the initial contraction had been removed. For the first few minutes after the convulsion the cortex was found to be completely unresponsive even to much stronger stimulation than had before been used. A gradual return of excitability of the motor cortex was demonstrated.

From these experiments it seemed that absinthe, in the period prodromal to the convulsion, produced a marked hyperexcitability of the motor cerebral cortex, mild stimuli giving an exaggerated motor response. This hyperexcitability could be demonstrated up to the time when the general convulsion began. Immediately after the convulsion the cortex was completely unresponsive; this soon passed off and a gradual return of irritability was seen.

There was a complete absence of the tonic phase of the convulsion when the animal was kept under light ether anesthesia. This was probably due to a lowered irritability of the cortical or subcortical mechanisms which are responsible for this phase.

In a number of animals the ether was reduced to a minimum during the convulsion so that the clonic convulsive movements of the extremities were marked. It was then found that stimulation of the motor cortex inhibited the convulsive movements of the contralateral legs. If all four extremities were twitching violently and the fore-leg area in the motor cortex stimulated, the oscillations of the contralateral fore-leg would gradually grow less violent until the extremity was quiet, although movements of the other extremities were still marked. In like manner the hind-leg movements could be controlled.

This furnishes a marked example of the inhibitory action of the motor cerebral cortex. In terms of the present experiment it probably means that the lower motor centers are hyperactive and have escaped to some extent from cerebral control. Sherrington has discussed the inhibitory function of the cerebral motor cortex. He points out that it is difficult to demonstrate since normally excitation is more clearly seen. Under the influence of strychnine, Sherrington found that the inhibitory action of the motor cortex was very definite. We therefore find that, during the convulsion, stimulation on an area in the motor cortex inhibited the clonic movements in the extremity controlled by this area.

DISCUSSION

The experiments recorded in this paper furnish material for a further discussion of convulsive phenomena and permit a correlation of previously reported results that seem at first to be of divergent significance.

Thus Pike and Elsberg (1925) removed the motor cerebral cortex of the cat on one or both sides and found that the minimal dose of absinthe required to produce a convulsion one to three weeks after the operation was greater than for normal cats. This observation has been confirmed in the present study. Absinthe was given to a number of cats from which definite large portions of the motor cortex had been removed from five to eleven weeks previously. The average minimal dose of the drug required to produce convulsions in these cats was one and a half times that of normal animals. It would seem then that the motor cortex has a definite part in the initiation of the convulsion.

But against this view may be urged the fact that, as Pike and Elsberg showed, the convulsion appears and is bilateral after the motor cortex has been removed on one or on both sides. After unilateral ablation of the cerebral cortex the convulsive movements are not as strong on the contralateral side and the cat often rolls to the injured side. But it was noted in the present experiments that the convulsion was in every case bilateral even though the whole cerebral hemisphere had been removed on one side.

It may be possible then that the drug does not act on the cerebral motor cortex at all but rather on lower motor centers. There are two arguments against this hypothesis. First it has been shown that after removal of the motor cortex the dosage of the drug must be increased in order to produce a convulsion. And again it has been demonstrated that after administration of drug the motor cortex becomes hyperirritable so that stimulation with a weak current produces a localized clonic convulsion that spreads over the whole contralateral side of the body and then to the ipsilateral side. It seems very likely that wormwood does not produce its effect by acting upon any particular portion of the brain or spinal cord but that, on the contrary, it makes the whole central nervous system hyperactive. Certainly it is difficult to believe that the motor cortex is alone responsible for clonic convulsions.

One of the most important observations from the standpoint of experimental epilepsy is concerned with the hypersensitivity of the central nervous system produced by small lesions. In this field the studies of Barbour and Abel (1910), and of Abel (1912) and Syz (1923) are of the utmost importance. Their work was done upon the frog. Later Dandy and Elman (1925) showed that convulsions in cats with small healed lesions of the motor cortex occurred with a dosage of the drug much smaller than that required in a normal animal. Lesions in other portions of the cerebral hemisphere or of the central nervous system also rendered the animals hypersensitive but were not as efficacious as lesions of the motor cortex.

Both of these observations were confirmed in the present study. Cats with small healed lesions of the motor area had convulsions on doses of the wormwood one-half the size required for normal animals, and similarly, after injury of tracts in the spinal cord the dosage of the

drug could be reduced from the normal and yet convulsions occurred. This observation seems of some importance as applied to a study of human epilepsy. For as Dandy and Elman suggest it is quite possible that congenital abnormalities or healed lesions of the central nervous system render the patient more susceptible to convulsions.

The return of the ataxia in animals with the posterior columns and spino-cerebellar fibers cut when under the influence of the drug seems a remarkable phenomenon. These two cats had shown after the operation marked incoördination which, after three weeks, had almost completely disappeared. But in the period preceding the convulsion this condition returned to a very marked degree. The reappearance of the ataxia was noted as long as fifteen months after the operation.

The most novel and therefore the most interesting phase of this study has to do with stimulation of the motor cortex after administration of absinthe. The findings recorded show for the first time that in the period of excitement preceding the convulsion the motor cortex is more irritable than normal. During the actual convulsion, on the other hand, stimulation of the motor cortex does not increase the violence of the convulsion. Thus stimulation produces an inhibition of muscular contraction in the extremity controlled by the area stimulated. It is clear that convulsions occur after removal of the motor cortex so that it must be assumed that lower motor centers are rendered hyperirritable by the drug. During the convulsion, stimulation of the cerebral motor cortex inhibits the activity of these centers. This is therefore a good demonstration of the inhibitory action of the cerebral motor cortex. The motor cortex is relatively non-responsive in the period immediately following the convulsion and the return to normal responsiveness is a gradual one.

SUMMARY

If large portions of the cerebral motor cortex of the cat were removed on one or both sides, the minimal dose of oil of wormwood required to produce a convulsion was one and a half times as great as in a normal animal. The convulsion was bilateral even after removal of the whole motor area on one side, although a weakness of the side contralateral to the lesion was apparent. After minute injury to the motor cortex, on the other hand, the cats became hypersensitive to the drug and con-

vulsions occurred after administration of one-half the normal dose. Injuries to the spinal cord also rendered the cats more responsive to the action of oil of wormwood. In each case several weeks elapsed between the operation and the further experimentation. The animals with the posterior columns and spino-cerebellar tracts of the spinal cord cut showed after the operation marked incoördination of the hind-legs which largely disappeared after three weeks. This ataxia returned in the period of excitement before the convulsion.

The motor cortex was also stimulated in cats before, during and after wormwood convulsions. The motor cerebral cortex was found to be hyperresponsive immediately before the convulsion. Contraction of the muscles of an extremity was not released on removing the stimulus but clonic movements continued which frequently spread to other parts of the body until a generalized convulsion occurred. During the convulsion stimulation of an area in the cortex did not excite but rather inhibited the convulsive seizure in the group of muscles controlled by this area so that clonic movements no longer occurred. The cortex was inexcitable even to strong currents for a period after the convulsion.

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THE HISTORICAL DEVELOPMENT OF THE PROCEDURE TERMED DÉBRIDEMENT

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The treatment of wounds in the World War has brought forth a number of tried and proven surgical precepts, some new, and some reapplications of old principles. Among the latter is the procedure called by the French *débridement*, a term for which no satisfactory English equivalent is as yet available. An impression has been prevalent that "its general application to wounds of war is new and represents one of the greatest advances made in traumatic surgery in many years" (1), but a review of medical biographies, and of such treatises on military surgery as that of Baron Larrey (2), offers definite evidence that débridement dates back to about 1790, and is based on a surgical principle first emphasized by a noted French surgeon, Pierre Joseph Desault.

In the early days of the recent war lessons from the past were overlooked, and contused and lacerated wounds caused by fragments of shell and shrapnel were treated by a variety of methods. For the most part "gunshot wounds when first received were treated conservatively and active surgical interference was postponed until infection had developed" (3). Wretched results and a grim necessity for a rapid convalescence forced the surgeons to seek improvement in the treatment of such wounds.

In November 1914, in a case of a superficial gutter wound, Colonel H. M. W. Gray (4), Consultant to the British Expeditionary Forces at Rouen, France, utilized a procedure of excision of the wounded and devitalized tissues together with removal of foreign bodies, which he called "Excision and Primary Suture." He usually employed local anesthesia. Iodine was introduced into every part of the wound and

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painted on the surrounding skin. With a sharp scalpel all lacerated tissue was then cut away *en masse*, one-third to one-half an inch from the raw surface. The wound was washed with saline solution and then closed with sutures to obliterate dead space as far as possible. Healing occurred by first intention, and much time was saved both in the period of hospitalization and in the necessary attention to the wound. After eight months' trial of the method, Gray was so impressed with its possibilities that he urged that it be tried whenever feasible in war wounds. Much credit is due him for its development, and within a year after the beginning of hostilities this treatment had found many advocates.

The term *débridement* was not applied to the method until after several years of warfare and after Depage (5) of Belgium and Lemaitre (6) of France had by their demonstrations and writings helped to establish the importance of excising damaged tissues. By 1917 débridement had become the standard and almost universal method in the allied armies of treating lacerated wounds due to projectiles.

An interesting analysis of this procedure and its application was made by Dean Lewis in 1919 (3).

> Excision of devitalized tissue has always been practiced to some extent in traumatic surgery. . . . . Two factors in the recent war made necessary the return to débridement which was advised and rather extensively employed by Larrey in the Napoleonic wars. These factors were, first, the high explosives, which, when they strike, tear and devitalize large pieces of muscle and shatter bone, and second, highly virulent bacteria which were present in the soil of the country over which the contending armies fought. This soil had been intensively fertilized for years.
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> In performing débridement an anatomic dissection should be done. The skin and subcutaneous tissues should be incised over the wound track and the tissues exposed by sharp dissection, down to tissue which is evidently devitalized. Devitalization is indicated by hemorrhagic infiltration of the muscle, and by failure to contract and bleed when pinched with tissue forceps or cut. Muscle, the seat of gas gangrene, has a peculiar waxy appearance and is rigid. It looks somewhat like muscle which has been boiled a short while. Muscle which is the seat of such changes should be entirely removed.

About a month after Gray first advocated this mechanical treatment of war wounds a publication by Morley (7), based upon experiences in the Gallipoli peninsula, emphasized the same idea; namely, that

complete excision of the soiled and devitalized tissues can be relied upon to secure healing by first intention.

The same principle had been presented by Martin Tinker (8) of Cornell, in 1909, in an article on compound fractures. As a more rational method of dealing with lacerated and contused wounds, he proposed thorough swabbing of the wound and adjacent skin with a powerful germicide, excision with the knife of tissues damaged by injury or by the germicide, and wound closure without drainage as in a clean incised wound.

In 1899 Pilcher (9) stated, in his book on the treatment of wounds, that primary cleansing of the wound with removal of foreign matter, together with detached fragments of bone and soft tissue is important in cases of contused and lacerated wounds. Tissues into which foreign matter have been ground should be trimmed away with the scissors or knife, and viable bruised portions of the tissue carefully cleansed and replaced. But in view of the fact that bruised wound surfaces have to struggle to retain their own vitality within the larger mass of partially devitalized tissue, he advised full provision for drainage.

Experiences in the Franco-Prussian and Russo-Turkish wars had led von Bergmann (10) to advocate conservative treatment in severely lacerated and avulsed wounds. In the battles of 1870-71 little was known of gunshot wounds from the anatomical point of view, and the seriousness of a wound was judged to depend mainly on the organ involved. The new projectiles tore the tissues deeply and the Russo-Turkish campaign was marked by an advance in methods of treatment based on the knowledge that infection constituted the major menace and that the effect on the organ or the bone was secondary. Von Bergmann felt that the primary surgical treatment should be limited to absolutely necessary details such as the removal of fragments, bone splinters, torn and soiled portions of skin and muscle, and the ligation of arteries, with the application of an antiseptic gauze dressing to the wound.

The antiseptic treatment of contused and lacerated wounds in Billroth's clinic was described by von Hacker (11) in 1883 (12). The surrounding parts were thoroughly cleansed with soap and water, shaved, and the wound freed from all foreign matter by the use of hot sublimate solution. All recesses and irregularities which might have been pene-

trated by foreign bodies were carefully explored. Devitalized or badly damaged tissue was cut away and all bleeding controlled by ligatures. Whenever possible, the wound was closed with continuous suture, provision being made for drainage. Otherwise, the wound was dusted lightly with iodoform and dressed with iodoform gauze.

Agnew (13) in his *System of Surgery* published in 1878, at the advent of the antiseptic era in surgery, felt that in gunshot wounds with much laceration of soft parts the best results followed irrigation with carbolized water with no attempt at primary closure.

Ferguson, in his book (14) on practical surgery, 1857, advised bringing a contused lacerated or gunshot wound into the condition of a simple incised one as far as circumstances would permit; urging the removal of foreign matter, and then closure. He maintained that when the devitalized area is large, separation can occur only by sloughing and that in these cases the wound must be allowed to suppurate.

During the war of the Rebellion (15) and the Crimean war (16) contused and lacerated wounds were cleaned, foreign matter was removed, and the wounds allowed to granulate. Nothing is said about débridement or of primary closure in the treatment of these wounds.

The earliest record of débridement, as the term is understood today, is to be found in the Memoirs of Military Surgery written in 1812 by Baron Larrey (2), military surgeon-in-chief to Napoleon. In the course of 53 years' service, Larrey (17) was with the French armies through 21 single and 5 double campaigns. His description of the procedure is given at the beginning of the chapter on the Campaigns of the Rhine. On his return from a voyage to Newfoundland and America and before the Rhine campaign, Larrey attended lectures given by Desault at the Hôtel Dieu. These talks were based in part on Desault's experiences in military surgery in 1789 to 1793 during the early part of the Revolution. Larrey summarizes Desault's principles as follows:

> 1. Spirits were used in the dressing of all gunshot wounds. Desault explained to us that their use was pernicious and that we should return to the emollients recommended and used by Ambroise Paré. Vegeto-mineral water (a solution of acetate of lead in water) had also been used.
>
> 2. Desault likewise showed us by examples that too extensive a débridement of these wounds produced muscular hernia.

3. It was a prevalent opinion that mere incisions changed the nature of gunshot wounds. Desault taught us that in order to change the nature of wounds from a complicated to a simpler state, it was not sufficient to make the part bleed; that the only means of accomplishing this was to refresh the contused edges of the wound with a sharp instrument and then to reunite the edges of the wound by means of a suture; and that this method was practicable only in the wounds of the face with laceration of the soft parts of the mouth. In my campaigns in Germany and Egypt I have put to profitable use the practical teaching of this man of genius who here appears to have made one of the most important and significant discoveries in all surgery.

This classical description was as impressive then as it is now. Ambroise Paré (1510-1590) (18), the barber surgeon who became a master of war surgery, had allowed his gunshot wounds to suppurate after pieces of cloth, bullets, parts of armour, splinters of bone, and bruised flesh and the like had been plucked forth.

In its early usage the word *débridement* meant an incision or unbridling. A French medical dictionary (19) of 1810 defines débridement as the action of cutting certain parts which, like a bridle, constrict or strangulate the organs which they cover. Probably the first appearance of débridement in an English dictionary is in the 1842 edition of Dunglison's medical dictionary (20) where it is defined as the removal of filaments in a wound or abscess which prevent the discharge of pus. A more general application is in the cutting of a soft membranous or aponeurotic part which interferes with the exercise of any organ whatever; thus, in paraphymosis débridement of the prepuce alleviates inflammation of the glans; in strangulated hernia débridement of the abdominal ring relieves the stricture of the intestines.

In describing wounds of the head in the Egyptian campaign, Larrey tells of a case which parallels in a remarkable way reports of the recent war wounds of the face and head. A corporal, Louis Vauté, received a bullet full in the face at the siege of Alexandria in 1801. The lower jaw and three fourths of the upper jaw were almost completely shot away. Destruction of the soft parts extended back as far as the second molar and also up to the temporal articulation of the jaw. The nasal bones as well as the ethmoids were crushed, and the zygoma and right eye injured beyond repair. Half of the tongue was cut away and the posterior nares and pharynx were widely open. A large laceration

of the neck exposed the jugular vessels. The patient had been given up for dead. Larrey found him and succeeded in getting the man to take some broth through an oesophageal tube. After his patient had gained sufficient strength he operated as follows: "The bruised and lacerated soft parts were excised after foreign bodies had been removed, and the great wound was scrupulously cleaned. Some vessels were ligated and the parts allowed to fall in position as much as possible, being maintained in apposition by various points of suture. Nourishment was given by the oesophageal tube." The wound did well and gradually closed. A silver mask was made to cover the deformity of the face.

During the World War a number of short articles (21, 22, 23) were published concerning Napoleon's surgeon general, Baron Larrey, in some of which passing mention was made of Desault's teaching concerning the excision of bruised tissues and primary suture, or débridement. Larrey in his memoirs had emphasized the use of salt solution in simple wounds and of Labarraque's solution in suppurating wounds. But no one seems to have given credit to his teacher, Desault, for the great principle of wound treatment which Larrey considered one of the most outstanding discoveries in surgical science.

In view of these facts we cannot justly claim that débridement in its general application to wounds of war is new and represents one of the greatest advances made in traumatic surgery in many years. A very comprehensive account of the life of this great master, Desault, whose teachings were given a wide practical application by Larrey, can be found in the eulogy written by his devoted pupil Bichat (24), and in the French medical biographical series (25) issued about 1822.

Pierre Joseph Desault (26, 27, 28) was born on the sixth of February, 1744, of parents who, though poor, gave their children a very good education. He first studied under the village priest and became proficient in literature and particularly in mathematics, later choosing the profession of surgery rather than entering the priesthood as his parents wished. He began with a country practitioner who was really of the barber-surgeon group. He then extended his medical studies to the military hospital at Béfort where his keen powers of observation obtained for him a valuable foundation in the principles of surgery. Three years later, in 1764, he went to Paris to study under the cele-

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brated surgeon Antoine Petit, devoting his time to anatomy, surgery, and to practical work in the hospitals, besides teaching mathematics to fellow students to increase his modest resources.

Following an illness from overwork, at the age of twenty-two he instituted a public course of anatomy in the winter of 1766, and soon had a following of three hundred pupils, most of them older than himself. Many of these came from afar, attracted by his clearness of demonstration, by the methodical arrangement of his descriptions, and especially by his indefatigable zeal as a teacher. Pupils deserted the lectures of the regular professors who became jealous; they demanded their rights and Desault's lectures were forbidden. Louis came to his rescue by engaging him as a teacher and showed his confidence by frequent attendance at his classes.

"He soon enlarged on the existing teaching of anatomy to a consideration of the form, magnitude, position and direction of the various parts of the body, emphasizing at the same time the characteristic pathology of these parts. In this way he created in France the first principles of surgical anatomy" (25). His objection to charts and wax models in teaching went so far that he wished to dispense with the dogmatic treatises then in general use, and substitute in their place careful studies of the cadaver by which the true picture of dissection would become engraved on the student's mind. His understanding of anatomy as applied to the surgical inadequacies of the day soon brought him from the first rank of anatomists to that of surgeon.

The impossibility of securing good union in fractures of the clavicle had been a surgical axiom since the days of Hippocrates. Desault, trained in mathematics, applied to this problem the principle of levers, using a wedge-shaped pad with the base up in the axilla to act as a fulcrum and fixing the arm with the elbow against the thorax. This method made it possible to counteract the weight of the shoulder and the downward and forward muscular pull so that the fracture was exactly reduced. A little later he devised a straight thin scalpel to replace the old curved and double-edged one used in amputations. He also re-introduced the immediate application of the ligature in wounds, forgotten since Paré's time.

As surgeon-in-chief of the Charité Hospital he worked out the treatment of fractures of the olecranon and patella and of dislocations of

the radius. Impressed by the success of Théden's pressure treatment for varicose ulcers he applied this principle to scirrhous ulcers of the rectum, using graduated tampons, and also successfully treated fistula in ano. He recognized and treated polyps of the oesophagus and improved upon Louis' treatment of harelip. In preference to the ineffectual pressure bandage he advocated ligation of the sac in umbilical hernia as done by the Greeks and Arabs.

In 1785, several months before John Hunter's description of the treatment of aneurysm, Desault ligated the artery in the lower thigh for a popliteal aneurysm, on the supposition that the pressure of this vascular tumor on the main artery was causing the gradual dilatation of the anastomosing vessels. Desault's idea seems to have been that this ligation proximal to the aneurysmal tumor would have a sparing effect on the collateral bed, and besides being feasible when other operative procedures were impossible, would lessen the danger of operation and of a serious aftermath. Hunter (29) had, however, already established by experiments "that the anastomosing vessels of the limb in their natural state would be capable of immediately taking on such increased action as would suffice for carrying on the circulation to the parts below the point at which the main artery was tied."

In 1785 when Desault was called to the Hôtel Dieu he was doing most of the major surgery in Paris. At this hospital he succeeded in establishing a clinical school for which an amphitheatre was erected.

Desault was married and had his home, yet he always slept at the Hôtel Dieu in order to be able to attend the sick at night. He was always the first to make rounds in the morning, and by eight o'clock was ready to go to the amphitheatre or to rejoin his pupils. According to Bichat, his clinics began with a public consultation to which charity patients were admitted. Desault took the history, pointed out the analogies of his observations with the patient's story, and after discussing the curative indications, outlined the treatment. A careful and detailed history of each of the interesting patients to be discharged that day was then read aloud by the student in charge, and discussed. These histories were composed of daily bedside notes and formed an accurate picture of the progress of each case. In instructing themselves in this way the pupils at the same time contributed to the experience of their comrades. The third and main part

of the lesson was devoted to operations, each one preceded by a discussion of the disease, the surgical indications, the probable outcome, and the measures which might serve to minimize the post-operative sequelae. The patient was then brought to the amphitheatre where Desault, assisted by the house surgeons, operated before all of his pupils. The various steps of the operation were discussed by the professor and the principles summarized, after which a conference was held on the progress of previous operative cases and of other patients in the hospital. The opening of cadavers followed. Autopsy studies were made for the instruction of the students, and the lesson ended with a dogmatic talk on some pathological point.

It was not until after this morning session, which usually lasted until noon, that Desault made his outside calls. He returned to the hospital at six, not to go out again until the following day. On his return he made a second complete round of the wards and then went to the amphitheatre for the evening lecture which was devoted to anatomy and to surgical procedures.

Jealousy still opposed him; his public operations were antagonistic to the religious beliefs of the Hôtel Dieu and alarmed its directors. The internes objected to the change in the old routine and to their added work. He, however, gradually overcame these obstacles and the teaching at the Hôtel Dieu was recognized as the best in France or in any of the established European schools. More than six hundred auditors, men from Italy, Spain, England, and Germany, came to learn from him a simplified logical basis for treatment and one that should be free from ancient prejudices. Truly a pupil of Zadig, observation was his sole teacher.

In one biography (25), written about a quarter of a century after his death, he is reproached because of his great prejudices against medicine. He ignored this science, disdained it, and repelled as false all ideas that referred to it. Besides refusing to study the progress of an internal disease he would never talk of one to his pupils. His prejudice extended even to those who were practicing or studying this art. He called them charlatans and doubted nothing so much as the reputation of an internist. Aside from this fault he held the admiration of all and was the greatest surgeon of Europe when the Revolution broke.

In 1791 he published his *Journal de Chirurgie* in which he described

the most interesting occurrences in his school and detailed the improvements he was trying to make.

He died June 1, 1795, believed by some to have been poisoned after attending the son of Louis XVI. This charge was denied by reputable men, and the autopsy performed by Bichat showed no evidences of poison.

To Desault, the investigator, the observer, the teacher, the first to establish clinical instruction in surgery, belongs the honor of having introduced one of the greatest advances in war surgery,—the mechanical disinfection of wounds, *débridement*.

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BOOK REVIEWS

Textbook on Diseases of the Skin and Syphilis. By ALBERT STRICKLER, M.D. Professor of Dermatology and Syphilology, Temple University of Medicine. 689 pp. (*F. A. Davis Company, Philadelphia, 1927.*)

This book contains 669 pages, which makes it about one-half the size of the usual standard text-book of skin diseases.

The author offers, as special features, the use of tables in presenting differential diagnosis, the indication of the purpose of each drug in the various formulae given and an attempt to classify the diseases of the skin, as far as possible, according to etiology. The first two are helpful in teaching and add some attraction to the volume. The last is a worthy ambition but at its best is too imperfect to have much value. The subject matter of the book for the most part is well presented, although some of the uncommon diseases are so briefly considered that the reader obtains little more than a mere enumeration. Some of the photographs are good while others are very poor. The attractiveness of the book would be increased if the few colored plates were left out.

L. W. K.

Practical Surgery of the Joseph Brice Hospital. By JAMES WILLIAM KENNEDY, M.D., F.A.C.S. 861 pp. $10.00. (*F. A. Davis Co., Phila., 1926.*)

This volume contains an account of the methods used and results obtained in the author's private hospital. With the exception of surgery of the breast the work is confined to gynecology and abdominal surgery.

Dr. Kennedy begins with a short biographical sketch of the late Dr. Joseph Price, who was his preceptor and the founder of the hospital. The author's expressed purpose is to make the volume a memorial to Dr. Price, whose methods he has followed, and whom he quotes liberally in every chapter. Indeed the work of hardly any other surgeon is mentioned. The work is a remarkable tribute of a student to his master, and shows everywhere the strong influence exerted by the master on the student.

The book is well and forcibly written. Dr. Kennedy has pronounced views, and expresses them with vigor. There is much, however, with which a great many would not agree. To give just one example, he never operates for breast tumors without removing the entire breast and dissecting the axilla. However, he never removes the pectoral muscles, cancer of the breast being no exception. There is much emphasis laid on "practical" considerations throughout the work, and perhaps a tendency to belittle scientific workers. Results are quoted in general terms rather than being based on statistical studies. One gets the impression from reading the book that the Joseph Price Hospital is a good "practical" hospital where dogma holds considerable sway.

A. R. K.

Practical Lectures on the Specialties of Medicine and Surgery. Delivered under the Auspices of The Medical Society of the County of Kings, Brooklyn, N. Y. Second Series, 1924–1926. 590 pp. $7.00. (*Paul B. Hoeber, New York.*)

This volume, the second in the series, contains lectures by thirty-seven different men, delivered before the Medical Society of King's County, Brooklyn, N. Y. The editors in a foreword state that the lectures were originally designed to bring to the attention of practising physicians information that was "most likely to be of practical value" to them. A very wide range of subjects is covered and one finds titles relating not only to general medicine and surgery, but also to the various specialties. Public health is represented and not even forensic medicine is overlooked. The subject-matter covered in this collection is of too wide a range to permit here of a comprehensive critical review, but the names of the lecturers are a sufficient guarantee of the worth of the material presented. The book is attractively printed and copiously illustrated.

A. M. C.

Preventive Medicine and Hygiene. Fifth Edition. By MILTON J. ROSENAU. 1458 pp. (*D. Appleton and Company, New York, 1927.*)

This treatise brings together, from a widely scattered and not always readily accessible literature, the most practical scientific information concerning public health procedures. No other publication, so far as I know, so thoroughly covers the entire field of science in its relation to hygiene and sanitation. In this edition the entire subject matter is brought up to date, and many of the topics have been entirely rewritten. The work is improved by a comprehensive treatment for the first time of the following subjects: psychoanalytic approach to sex hygiene, the prevention of cancer, the conservation of vision, periodic physical examinations, flukes and their relation to disease, granuloma inguinale, balantidial dysentery, resuscitation, gas masks, statistical methods and practical points in public health administration. Students of medicine will welcome the new edition as a most valuable work of reference.

R. R. H.

Lehrbuch der Physiologie. By EMIL ABDERHALDEN. Four volumes. Vol. 1, 18 marks; Vol. 2, 24 marks; Vol. 3, 33 marks. (*Urban & Schwarzenberg, Berlin u. Wien, 1925–1926.*)

The four large volumes of this excellent work constitute a valuable addition to the library of medical science. The indefatigable author, who several years ago gave us the well known "Lehrbuch der physiologischen Chemie" and the encyclopedian revue "Handbuch der biologischen Arbeitsmethoden," stresses in the present work the specific coördination in the relation between structure and activity of the various organs and the important relationship between the parts of the organism. That human physiology and anatomy are indispensable in practical medicine extends like a red thread throughout his whole presentation of physiol-

ogy. Special chapters are devoted to the discussion of coördination and regulation of activities of the organs. From such knowledge there can already be seen to some extent where failure in the struggle for normal functional achievements is liable to occur.

The comprehensive character of the labor which Abderhalden has undertaken and the admirable way in which he appears to have been executing it forbids any pedantic criticism. The only criticism I could make is that the essayist calls his presentation of the whole subject "fragmentary work."

An immense literature has been drawn upon. He not only knows the literature, but, a more valuable attribute, he understands how to make use of the literature. Indeed, he not infrequently enjoys leaving us with more problems than he sets out to solve. In addition, the author quotes foreign literature as largely as that of his own country. As an example, in the most attractive chapters dealing with the ductless glands, he cites on several occasions the excellent researches of Abel and co-workers.

The judicious appraisal the reviewer extends to the whole work culminates in the recommendation that the book well merits translation, thus making it available in the English language to a large body of readers.

J. H.

The Prevention of Dental Caries and Oral Sepsis. By H. P. PICKERILL, C.B.E., M.D. Third edition. 340 pp. $6.00. (*Paul B. Hoeber, New York, 1924.*)

"The Prevention of Dental Caries and Oral Sepsis" is far-reaching in its exhaustive research into the various causes that produce the most general and disastrous disease of the human denture. In the historical discussion Dr. Pickerill points out the few means for the prevention of dental caries that are emphasized today. His chapter on the incidence of this disorder gives much comparative information, and includes the findings relative to the dentures of the available skulls of the principal museums of the world. It is evident that this disease has progressed and accompanied civilization in proportion to the degree attained by the various races.

The results of the pathological findings show that there exists no great difference between the oral flora of immune and that of susceptible individuals.

Diet, habit and heredity are all considered in their influence in lowering the resistance to caries. The chapter on the protective organ of the tooth, the enamel, should be especially instructive to the dental student.

The oral secretions are dealt with at length. The conclusions in reference to saliva are that it provides a natural and potentially perfect mouth wash, which acts continuously; that it is completely under the control of the individual; that it may be altered or varied in amount or composition; that its beneficial effects may be increased or decreased at will.

Much space is given to the various factors producing caries such as the different diets used by the nations and tribes of the world.

Pickerill suggests legislative measures to regulate the food supply so that only the proper kinds may form the principal diet.

In conclusion, he states that the preventive treatment of dental caries must be made one of the most important branches of public health service. To initiate and organize a department to deal efficiently with this branch alone would be (he thinks) a source of national economy.

W. H. B.

The Significance of the Physical Constitution in Mental Disease. By F. I. WERTHEIMER and FLORENCE E. HESKETH. Medicine Monographs, vol. X. 76 pp. $2.50. (*The Williams & Wilkins Co., Baltimore, 1926.*)

The last few decades have produced an enormous number of good as well as of worthless investigations appertaining to the physical constitution of man. The bulk of this literature consists of French and German publications. The particular study by Wertheimer and Hesketh, which constitutes a sound and definite contribution to the problem of the correlation between mental disease and constitution, is certain to stimulate and influence other English-speaking students interested in this highly important and comparatively recent field of science. A part of this book is devoted to a valuable and practically complete historical review of the various classifications of body types. Another and very considerable part deals with the detailed observational and metric methods of determining morphological types. A carefully chosen and accurately explained technique is most essential in pioneer work of this sort which strives to replace the vague general impressions of the older authors by precise, impersonal figures. The results obtained by these technical procedures are exhaustively and critically analyzed in the last part. The most significant proportion, and one which can easily be put to clinical use, was found to be the relation between limb length and trunk volume. The many other well-justified conclusions do not readily lend themselves to a brief summary. If some of these results may as yet have to be regarded as being of a more or less tentative nature, this is entirely due to the fact that research on constitution problems has so far been rather one-sided. Indeed, much more work has been done in regard to the relation between constitution and pathology than on some of the most fundamental problems of normal constitution which still await sober investigation in place of the many recent, unfounded speculations. The science of "constitution" represents a field of biology in which the domains of the geneticist, the anthropologist, the physiologist, and the clinician overlap and in which close coöperation of all these students is absolutely necessary for further progress.

A. H. S.

BOOKS RECEIVED

Modern Medicine. Its Theory and Practice in Original Contributions by American and Foreign Authors. Edited by Sir William Osler, Bart., M.D., F.R.S Third edition, thoroughly revised. Re-edited by Thomas McCrae, M.D. Assisted by Elmer H. Funk, M.D. Volume IV. Diseases of the Respiratory System—Diseases of the Circulatory System. Illustrated. 1927. 8°. 1011 pp. Lea & Febiger, Philadelphia. $9.00.

Text-Book of Bacteriology. By WILLIAM W. FORD, M.D. Illustrated. 1927. 8°. 1069 pp. W. B. Saunders Co., Philadelphia.

Surgical Anatomy of the Human Body. By JOHN B. DEAVER, M.D., Sc.D., LL.D., F.A.C.S. Second edition. In three volumes thoroughly revised and rearranged. Profusely illustrated with drawings from original dissections made especially for the work. Volume II. Upper Extremities, Neck, Shoulder, Back, Lower Extremities. Volume III. Joints of the Lower Extremities, Chest, Thorax, Abdomen, Pelvis, Perineum. 1927. 4°. 854 pages; 763 pp. P. Blakiston's Son & Co., Philadelphia.

Lectures on Internal Medicine. Delivered in the United States, 1926. By KNUD FABER, M.D. With forty-three figures and charts. 1927. 8°. 147 pp. Paul B. Hoeber, New York. $3.00.

International Clinics. A Quarterly of Illustrated Clinical Lectures and Especially Prepared Original Articles. Edited by Henry W. Cattell, A.M., M.D., with the collaboration of Chas. H. Mayo, M.D., Thirty-seventh series, vol. 2. 1927. 8°. 308 pp. J. B. Lippincott Company, Philadelphia.

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Practical Otology. By MORRIS LEVINE, M.D. Illustrated with 145 engravings and 3 colored plates. 1927. 8°. 387 pp. Lea & Febiger, Philadelphia. $5.50.

Clinical Physiology (A Symptom Analysis) in Relation to Modern Diagnosis and Treatment. A Text for Practitioners and Senior Students of Medicine. By ROBERT JOHN STEWART MCDOWALL, D.Sc., M.B., F.R.C.P. (Edin.). With an introduction by W. D. Halliburton, LL.D., F.R.C.P., F.R.S. 1927. 8°. 383 pp. D. Appleton and Company, New York. $7.00.

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Pioneer Medicine in Western Pennsylvania. By THEODORE DILLER, M.D. With

a foreword by J. J. Buchanan, M.D. With twenty-six full page illustrations. 1927. 12°. 230 pp. Paul B. Hoeber, New York. $3.00.

Overcoming Tuberculosis. An Almanac of Recovery. By GERALD B. WEBB, M.D. and CHARLES T. RYDER, M.D. Third edition revised. 1927. 12°. Paul B. Hoeber, New York. $2.00.

How to Make the Periodic Health Examination. A Manual of Procedure. By EUGENE LYMAN FISK, M.D., and J. RAMSER CRAWFORD, M.D. Foreword by Major General Merritte W. Ireland. 1927. 8°. 393 pp. Macmillan Company, New York. $4.00.

City Health Administration. By CARL E. MCCOMBS, M.D. 1927. 8°. 524 pp. Macmillan Company, New York. $5.50.

The Psychology of Mental Disorders. By ABRAHAM MYERSON, M.D. 1927. 16°. 135 pp. Macmillan Company, New York. $1.40.

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Evolution of Preventive Medicine. By Sir Arthur Newsholme, K.C.B., M.D. F.R.C.P. 1927. 16°. 226 pp. Williams & Wilkins Company, Baltimore. $3.00.

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Archives of Neurology and Psychiatry. From the Central Pathological Laboratory of the London County Mental Hospitals, Maudsley Hospital, Denmark Hill. Edited by Frederick L. Golla, F.R.C.P., M.B. (Oxon), vol. ix. 1927. 8°. Published by the London County Council, London.

The Hepatic Principle, Anabolin, Detoxication by the Liver and the Control of Functional Hypertension. By Henry R. Harrower, M.D. With a foreword by Ivo Geikie Cobb, M.D. And an historical introduction by Victor Robinson, M.D. 1927. 8°. 147 pp. Baillière, Tindall and Cox, London.

Diagnosis and Treatment of Diseases of the Stomach. With an Introduction to Practical Gastro-Enterology. By Martin E. Rehfuss, M.D. With 519 illustrations, some in colors. 1927. 8°. 1236 pp. W. B. Saunders Company, Philadelphia and London. $12.00.

Physical Diagnosis. By Richard C. Cabot, M.D. Ninth edition. Revised and enlarged, with six plates and 279 figures in the text. 1927. 8°. 536 pp. William Wood and Company, New York. $5.00.

Manual of the Diseases of the Eye. For Students and General Practitioners. By Charles H. May, M.D. Twelfth edition, revised. With 374 original illustrations including 23 plates, with 73 colored figures. 1927. 8°. 445 pp. William Wood and Company, New York. $4.00.

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Malarial Psychoses and Neuroses. With chapters Medico-Legal, and on History, Race Degeneration, Alcohol, and Surgery in Relation to Malaria. By William K. Anderson, M.D., F.R.F.P.S. (Glas.). 1927. 8°. 395 pp. $13.00.

An Illustrated Guide to the Slit-Lamp. By T. Harrison Butler, M.A., D.M. (Oxon.), M.R.C.S. (Eng.), L.R.C.P. (Lond.). 1927. 8°. 144 pp. $9.00.

The Health of the Child of School Age. By various authors. With a foreword by Sir Thomas Oliver, M.A., M.D., F.R.C.P., LL.D., D.C.L. 1927. 12°. 204 pp. $1.80.

Manual of Bacteriology. By ROBERT MUIR, M.A., M.D., Sc.D., LL.D., F.R.S. and the Late JAMES RITCHIE, M.A., M.D., F.R.C.P. (Ed.). Revised with the co-operation of Carl H. Browning, M.D., D.P.H. and Thomas J. Mackie, M.D., D.P.H. Eighth edition. With 211 illustrations in the text and 6 coloured plates. 1927. 12°. 821 pp. $4.75.

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